al blood product transfusions given as required, was clinically successful.

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Reply to the Editor:

Lemmer raises a number of interesting points with regard to the removal of abciximab (ReoPro) during cardiopulmonary bypass with the use of a hemoconcentrator.[1]

First, as correctly pointed out, the experiments were initially carried out with normal saline solution, as a proof of principle. However, as stated in the article, the experiments were repeated with the use of packed red cells to maintain clinical relevance. As the free abciximab is biologically active (Eli Lilly and Company data sheet), then our protocol, even though simplified, was clinically relevant.

Second, with regard to analyzing native platelet function, platelet dysfunction after cardiopulmonary bypass occurs for a myriad of known and unknown preoperative and perioperative reasons, making the evaluation of an intervention difficult. The aim of this study was to demonstrate a technique to reduce the inhibition of transfused platelets that occurs as a result of abciximab inactivation. This should result in a reduction of postoperative platelet transfusion requirements and transfusion-related complications, for example, adult respiratory distress syndrome. We make no claims to reversal of the effect of abciximab on native platelets. However, by implication from our results and the fact that abciximab binding is known to be a reversible process, hemofiltration will result in at least a partial reversal of the effect of abciximab on native platelets.

Third, Lemmer states that the dissociation of abciximab from platelets is slow, but he then states that abciximab redistribution from native platelets to transfused platelets occurs immediately after their administration, a kinetics contradiction. Either a platelet-to-platelet interaction occurs (microaggregation, which is known not to be the case) or free abciximab is involved. It is this free abciximab that Lemmer quite rightly implicates in causing transfused platelet dysfunction, which we demonstrated can be reversed.

Care should be taken with regard to interpretation of percentage glycoprotein IIb/IIIa receptor binding as evaluated in reference 3 provided by Lemmer, since no platelet function tests were performed, just fluorescence-activated cell sorter analysis, which does not necessarily correlate with physiologic function. The prior administration of aspirin or clopidogrel, for example, will result in an uninterpretable result in this situation.

In addition to abciximab (ReoPro), tirofiban (Aggrastat) and eptifibatide (Integrilin) are also specific glycoprotein IIb/IIIa inhibitors. The possible reversal of bleeding caused by glycoprotein IIb/IIIa inhibition therapy via hemofiltration is now alluded to by the drug information data sheets produced by Merck, which manufactures Aggrastat, and COR Therapeutics, Inc, and Key Pharmaceuticals, Inc, which manufacture Integrilin.

Unfortunately, we do not have access to the publication of Lemmer's group (in press). It would be interesting to note what degree of preoperative platelet inhibition was present in their patients, since it is widely known that abciximab has a variable clinical effect. Conventional platelet function tests are impractical in an emergency situation. However, we have recently demonstrated a quick, simple test of platelet inhibition caused by abciximab, via the technique of whole blood microaggregation, which involved the use of a standard laboratory Coulter counter (Coulter Electronics, Inc, Hialeah, Fla).[3] This should aid in assessing preoperative platelet inhibition, thus making it possible to rank patients by their susceptibility to bleeding, so that accurate evaluation of any antibleeding strategy can be ascertained.

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